Exhibit 19

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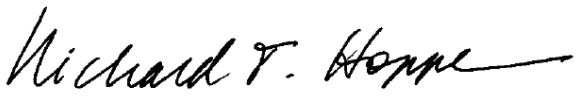

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- Depositions of:
  - Allan Wayne Howard dated 2/16/2024 (Plaintiff)
  - Elizabeth Howard dated 5/7/2024 (daughter)
  - James Pieckenbrock dated 5/7/2024 (friend)
  - Dr. [redacted]
  - Dr. [redacted]
  - Dr. [redacted]
  - David A. Savitz, PhD, dated 7/17/2024
- The Expert Report of Morris Maslia dated 10/25/2024
- The Expert Report of Dr. Kelly Reynolds
- The General Causation Reports of:
  - Timothy M. Mallon, M.D., M.P.H., MS.
  - Dean W. Felsher, M.D., Ph.D
  - Kathleen Gilbert, Ph.D
  - Steven B. Bird, M.D
  - Howard Hu M.D. M.P.H. Sc.D
- Short Form Complaint filed on behalf of Allan Wayne Howard on 7/15/24

In addition, I relied upon the peer-reviewed scientific literature that, in my opinion, is the most rigorous and relevant to the issues inherent in this evaluation. As appropriate, such evidence will be cited during this report.

III. Summary of Opinion

It is my opinion that it is more likely than not that Mr. Howard's non-Hodgkin lymphoma (NHL), diffuse large B-cell lymphoma (DLBCL),was caused by his exposure to the contaminated water at Camp Lejeune.

I begin with a discussion of the chemicals in the water at Camp Lejeune, briefly turn to their causative effect of NHL generally, and then to the differential diagnosis etiology of Mr. Howard's NHL.

IV. Chemicals at Camp Lejeune

Based on reports and testing, the water at Camp Lejeune contained TCE, PCE, benzene and the byproducts of their degradation.

a. TCE

Trichloroethylene (TCE) is an industrial solvent that has been widely used in various applications, including degreasing metals and in the production of adhesives and paints. TCE has a biologic half-life of three days. In humans, it is metabolized to trichloroepoxyethane (TCE oxide), then to trichloroacetaldehyde, chloral hydrate and other metabolites including trichloroacetic acid, dichlorovinyl glutathione, and dichlorovinyl cysteine. Some of these metabolites may be more toxic than the parent compound.

Epidemiological studies indicate a link between TCE exposure and the development of non-Hodgkin lymphoma (NHL). Lymphoma is a cancer that affects the lymphatic system, a crucial part of the immune system. There are several mechanisms through which TCE may contribute to the development of lymphoma.

TCE is a genotoxic agent (a property of chemical agents that damages the genetic information [DNA] in a cell)[1] via both direct and indirect effects on the DNA. It may cause chromosome aberrations, chromosome breaks, and sister chromatid exchanges.[2,3] This genetic damage can lead to mutations that contribute to the development of lymphoma

TCE has been demonstrated to cause immune system dysfunction.[4]. It has been shown to have immunotoxic effects, potentially altering immune function and leading to an increased risk of lymphoproliferative disorders, including lymphoma. Evidence from animal studies indicates that TCE exposure causes immunomodulation including autoimmune disease and immunosuppression.[5] Both autoimmune disease and immunosuppression are associated with NHL.[6] Studies conducted of Chinese factory workers exposed to TCE have observed alterations in immune function markers that have been associated with an increased risk of NHL, indicating that the associations observed between TCE and NHL are biologically plausible.[7] In another study of the cohort of Chinese factory workers, total lymphocyte counts decreased with increasing exposures to TCE. Similar exposure-response trends were observed for CD4+ T cells, CD8+ T cells, B cells and NK cells.[8] The study concluded that these results provided evidence that TCE exposure leads to immunosuppression, which is associated with an increased risk of NHL.[8]

Karami et al. conducted a meta-analysis of TCE exposure and risk of lymphatic and hematopoietic cancers.[9] They examined studies published between 1950 and 2011.[9] The meta-analysis for NHL included 293 NHL cases from 12 cohort studies and 8140 cases from 12 case-control studies.[9] Their conclusion was that the data supported as association between TCE exposure and increased risk of NHL (relative risk = 1.32, 95% confidence interval 1.14-1.54).[9] Scott and Jinot conducted another systematic review of the epidemiologic evidence for an association between TCE exposure and NHL.[10] They calculated a relative risk for developing NHL following TCE exposure to be 1.23 (95% CI 1.07-1.42) and for the highest exposure group to be 1.43 (95% CI 1.13-1.82).[10]

b. Benzene

Benzene is a colorless, toxic chemical compound that is widely recognized as an environmental and occupational hazard. It is primarily used in the manufacture of chemicals, plastics, and synthetic fibers. Benzene exposure has been implicated as a causative agent in the development of NHL.

Research indicates that benzene is a hematotoxic agent,[11] meaning it can adversely affect the blood-forming organs, including the bone marrow. This toxic effect can lead to disruptions in the production of blood cells, including lymphocytes, which are crucial components of the immune system. Epidemiological studies have consistently demonstrated an association between benzene exposure and an increased risk of developing various hematological malignancies, including NHL.

There are several possible mechanisms by which benzene contributes to lymphomagenesis. Benzene metabolites can induce genetic mutations, compromise immune function, and promote inflammation, all of which may lead to malignant transformation of lymphocytes. Multiple studies show that it produces genotoxicity in the lymphocytes of exposed humans.[12] It may produce multiple cytogenetic abnormalities in lymphocytes, and it induces specific chromosomal changes associated with NHL in human lymphocytes, The immunosuppression induced by benzene may lead to decreased immunosurveillance. In a recent study of the cohort of Chinese factory workers, benzene exposure was associated with alterations in lymphoid cell types and B-cell activation markers indicative of immunosuppression that could result in an increased risk of NHL.[8] Chronic exposure to benzene is known to result in genetic and epigenetic alterations that enhance lymphocyte proliferation and survival, further contributing to the development of lymphoma.

Benzene has also produced lymphomas in animal studies.[12] Accordingly, there is considerable support for the realization that it can cause human lymphatic tumors.[12] Linet at al. conducted a large study of mortality among benzene-exposed workers in China.[13] They compared causes of mortality in 73,789 benzene-exposed workers with 34,504 non-exposed workers in 12 cities in China.[13] The benzene-exposed workers experienced increased risk for all-cause mortality.[13] Notably, the relative risk for NHL was 3.9 (95% CI 1.5-13).[13] In a large meta-analysis of human studies, Rana et al., reviewed 20 case-control and eight cohort studies that included 9587 patients with NHL.[14] They reported increases in the risk for a wide variety of lymphomas and specifically a doubling of the risk for diffuse large B-cell lymphoma.[14]

c. PCE

Tetrachloroethylene (PCE) is a colorless, non-flammable liquid used for dry cleaning and as a metal degreasing solvent. It is regarded as a toxic substance, a human health hazard, and an environmental hazard. Numerous toxicology agencies regard it as a carcinogen.[15]

A study conducted in four Nordic countries found that high exposure to PCE was associated with an elevated hazard ratio for NHL of 1.23 (95% CI 1.00-1.52).[16] Furthermore, in a long-term mortality study of aircraft manufacturing workers, Boice et al. found an increased standardized

mortality rate of 1.70 (95% CI 0.73-3.34) for workers exposed to PCE.[17] In a long term follow up of the same study cohort, Lipworth et al. defined a standardized mortality ratio of 1.43 (1.00-1.98) related to PCE exposure and the risk for developing non-Hodgkin lymphoma.[18] Thus, the scientific literature supports an association between occupational PCE exposure and NHL.

V. ATSDR

In light of the test results of the water at Camp Lejeune the Government conducted a number of studies of the water. The leading study is what is known as the ATSDR report (ATSDR).

The ATSDR Report, or more fully, the "ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases"[19] published January 13, 2017, reviewed epidemiological studies involving TCE and PCE exposure conducted by the EPA,[20] IARC,[21] and NTP[22]; meta-analyses conducted by NCI researchers,[9] EPA,[10] and an IARC workgroup[16] for TCE and hematopoietic cancers. ATSDR utilized these reviews and meta-analyses to identify epidemiological studies for TCE and PCE. Meta-analyses of benzene and hematopoietic cancers[23,24,25] were used to identify epidemiological studies for benzene. In addition, literature searches using PubMed were conducted to identify epidemiological studies conducted after the meta-analyses and reviews were completed.

The ATSDR classified the evidence between exposure to the chemical agent and the development of cancer as "sufficient evidence for causation," "equipoise and above evidence for causation," "below equipoise evidence for causation," and "evidence against a causal relationship."[19] "Sufficient evidence" was further defined as sufficient evidence from human studies in which chance and biases (including confounding) can be ruled out with reasonable confidence, **or** there is less than sufficient evidence from human studies but sufficient evidence in animal studies and strong evidence that the agent acts through a relevant mechanism in humans.[19] Sufficient evidence from human studies could be provided by a meta-analysis and/or by several studies considered to have high utility.[19] Considerations with respect to the quality of the evidence included temporal relationship, consistent positive associations (e.g., risk ratio or odds ratio greater than 1.1), magnitude of the effect estimate, exposure-response relationship, and biological plausibility.[19]

"Equipoise and above" evidence implied that the evidence was sufficient to conclude that a causal relationship was at least as likely as not, but not sufficient to conclude that a causal relationship existed.[19] For example, if the degree of evidence from human studies was less than sufficient but there was supplementary evidence from animal studies and/or mechanistic studies that supported causality, **or** a meta-analysis did not provide convincing evidence (e.g., the summary risk estimate was close to the null value of 1.0, i.e., ≤1.1), or if the meta-analysis observed a non-monotonic exposure-response relationship) but there was at least one epidemiological study considered to be of high utility occurring after the meta-analysis had been conducted, in which an association between the exposure and increased risk of the disease of interest had been found and in which chance and biases could be ruled out with reasonable confidence, **or** a meta-analysis has not been conducted, but there was at least one epidemiological study considered to be of high utility in which an association between the

exposure and increased risk of the disease of interest had been found and in which chance and biases could be ruled out with reasonable confidence.[19]

The 2017 ATSDR report concluded, based upon its review, that there was sufficient evidence for causation between TCE exposure and the development of NHL, equipoise and above evidence for causation between PCE and the development of NHL and sufficient evidence for causation between benzene exposure and the development of NHL.[19] The data from the ATSDR Reports combined with the meta-analyses related to TCE exposure[9,10] provide compelling evidence that TCE exposure increases the risk for developing NHL. The ATSDR reports combined with the cohort study of Linet et al (2015)[13] provide a similar degree of evidence for the relationship between benzene exposure and NHL.

Based upon my years of experience, I agree with the ASTDR's definition of "at least as likely as not" or "equipoise and above." I also agree that TCE, PCE, and benzene all cause NHL, at least as likely as not.

VI. General Causation Reports of Drs. Felsher, Hu, Gilbert, Mallon, and Bird

I have reviewed and considered the general causation reports of Drs. Felsher, Hu, Gilbert, and Bird. Based on my background, education, and experience, the reports of these experts are robust and reliable. All have concluded, as do I, that the contaminants in the Camp Lejeune water supply were sufficient to cause NHL. See, for instance, Felsher Report, p. 37-40; Gilbert Report, p.31-35.

VII. Concentrations of Contaminants at Camp Lejeune

I have reviewed the expert report of Morris Maslia, dated October 24, 2024. Based on this report, Appendix H1 in particular, the concentrations of TCE were well in excess of 100 micrograms per liter of water during most of Mr. Howard's stay at Camp Lejeune, peaking at 546 micrograms per liter in December 1978. The MCL for TCE is 5 micrograms per liter. For PCE, levels were well above the MCL of 5 micrograms per liter for the vast majority of Mr. Howard's time on base. It peaked at 24 micrograms per liter – months in November and December of 1978.

I also have reviewed the expert report of Dr. Kelly Reynolds regarding the likely cumulative amounts of TCE, PCE and benzene that Mr. Howard ingested during his time at Camp Lejeune. Considering his days on base and cumulative contaminant exposure concentrations, and based upon his deposition-based informed activities, his cumulative consumption (total µg=days x concentration per deposition exposure assumptions) for TCE ranged between 660,782 ppb ingested to 1,019,982 ppb ingested (depending on assumptions of ingestion), for PCE it was 27,780 ppb ingested to 42,882 ppb ingested (depending on assumptions of ingestion), and for benzene it was 7,859 ppb ingested to 12,132 ppb ingested (depending on assumptions of ingestion).